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BROMOCRIPTINE IN PERIPARTUM CARDIOMYOPATHY: EVIDENCE, CONTROVERSY AND PRACTICAL USE

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ABSTRACT

Background

Peripartum cardiomyopathy is a potentially life threatening form of heart failure that develops mainly towards the end of pregnancy or during the months following delivery, pregnancy termination, or miscarriage. Cleavage of full length prolactin into an antiangiogenic 16 kDa fragment is one proposed mechanism of myocardial injury and pro-

vides the rationale for prolactin suppression with bromocriptine. However, the clinical efficacy and safety of bromocriptine remain uncertain, and European and United States recommendations differ.

Aim

To critically evaluate current evidence concerning the efficacy, safety, and practical use of bromocriptine in peripartum cardiomyopathy.

Materials and methods

A narrative review of English language publications issued from January 2000 through June 2026 was conducted using PubMed, the Cochrane Library, and Google Scholar. The final literature search was conducted in June 2026. Eligible sources included pre-clinical studies, clinical trials, observational studies, registries, systematic reviews, meta analyses, narrative reviews, clinical guidelines, position statements, and relevant editorials. A total of 44 sources were included in the narrative synthesis.

Results

Available clinical evidence suggests that bromocriptine may improve left ventricular ejection fraction, although benefits for major clinical outcomes remain uncertain. In the EORP PPCM Registry, the composite outcome occurred in 22% of patients receiving bromocriptine and 33% receiving standard care ($p = 0.044$). The association remained significant after adjustment (OR 0.47, 95% CI 0.31 to 0.70; $p < 0.001$), while thromboembolic event rates were similar. Small trials, treatment heterogeneity, and observational data preclude causal conclusions. European guidelines state that bromocriptine may be considered, whereas the United States guideline assigns no formal recommendation class.

Conclusions

Bromocriptine may be considered as an adjunct to standard heart failure therapy in selected women with peripartum cardiomyopathy, but its effects on major clinical outcomes remain uncertain. When bromocriptine is used, concomitant anticoagulation at a minimum prophylactic dose should be considered according to applicable recommendations and individual thromboembolic and bleeding risks. The consequences of lactation suppression should be discussed with the patient. Further adequately pow-

ered randomized trials are required to determine clinical efficacy, safety, optimal treatment duration, and appropriate patient selection.

Keywords: peripartum cardiomyopathy, bromocriptine, prolactin, heart failure, anticoagulation, pregnancy

1. INTRODUCTION

Peripartum cardiomyopathy also known as postpartum cardiomyopathy, is an uncommon form of heart failure that develops towards the end of pregnancy or within the months following delivery, termination or miscarriage in women without other identifiable causes for heart failure [1]. Worldwide incidence of PPCM is 1 in 2000 births [2] but it varies geographically. Africans and African Americans are at greater risk for developing PPCM than Caucasian population [3]. Pathophysiology of PPCM is complex and involves a combination of systemic angiogenic imbalance and host susceptibility, including genetic predisposition, oxidative stress, and anti-angiogenic 16 kDa prolactin. Risk factors for PPCM include: African descent, hypertension, preeclampsia, advanced maternal age, multigestational pregnancies and genetic factors [1]. Diagnostic criteria include reduced left ventricle ejection fraction (LVEF < 45%) with or without left ventricle dilation, onset during the peripartum period or in the months following delivery, and exclusion of other causes of heart failure [2].

PPCM contributes significantly to maternal morbidity. Recent discoveries about the harmful activity of 16 kDa prolactin reveal a promising therapeutic target in the treatment of peripartum cardiomyopathy. Peripartum cardiomyopathy (PPCM) is a clinically significant condition because it can lead to severe heart failure, life-threatening complications, and maternal mortality in otherwise healthy young women [44]. Early recognition and appropriate management are crucial, as timely treatment can significantly improve cardiac recovery and long-term outcomes. This uncertainty is reflected in differences between European and United States recommendations regarding patient selection, treatment regimens, and concomitant anticoagulation [2,22].

Aim

The aim of this narrative review was to critically evaluate current evidence on the efficacy, safety, and practical use of bromocriptine in peripartum cardiomyopathy.

Objectives

1. To examine the pathophysiological rationale for prolactin inhibition in peripartum cardiomyopathy.
2. To assess evidence from preclinical studies, clinical trials, registries, systematic reviews, and meta analyses concerning the efficacy and safety of bromocriptine.
3. To compare European and United States recommendations and discuss patient selection, dosing, anticoagulation, lactation suppression, monitoring, and shared decision making.

2. MATERIALS AND METHODS

Study Design

This narrative review summarized and critically evaluated evidence concerning the pathophysiological rationale, efficacy, safety, and practical use of bromocriptine in peripartum cardiomyopathy. Evidence from preclinical studies, clinical trials, observational studies, registries, systematic reviews, meta analyses, narrative reviews, clinical guidelines, position statements, and relevant editorials was synthesized narratively.

Search Strategy

A literature search was performed in PubMed and the Cochrane Library and was supplemented by Google Scholar. The final literature search was conducted in June 2026. The search covered publications written in English and published from January 2000 through June 2026.

The search terms included “peripartum cardiomyopathy,” “PPCM,” “pregnancy associated cardiomyopathy,” “postpartum cardiomyopathy,” “bromocriptine,” “prolactin,” “16 kDa prolactin,” “oxidative stress,” “genetic susceptibility,” “TTN,” “angiogenic imbalance,” “angiogenesis,” “clinical guidelines,” “anticoagulation,” “thromboembolism,” “lactation,” “breastfeeding,” “dosing,” “treatment regimen,” and “monitoring.” Keywords and relevant Medical Subject Headings were combined using Boolean operators in PubMed. Corresponding combinations of keywords were used in the Cochrane Library and Google Scholar. Reference lists of relevant publications were screened to identify additional sources.

Eligibility Criteria

Publications written in English were eligible if they addressed the pathophysiological rationale, efficacy, safety, or clinical use of bromocriptine in peripartum cardiomyopathy. Eligible sources included preclinical studies using relevant animal models, clinical trials, observational studies, registries, systematic reviews, meta analyses, narrative reviews, clinical guidelines, position statements, and relevant editorials. Clinical guidelines and position statements were included to compare European and United States recommendations concerning bromocriptine.

Publications were excluded if they were published before January 2000, were not written in English, or did not provide information relevant to the predefined scope of the review. A total of 44 sources were included in the narrative synthesis.

3. RESULTS

3.1 Pathophysiology of PPCM

The pathophysiology of PPCM is uncertain. Current evidence supports an interplay between oxidative stress, hormonal factors, angiogenic imbalance, and genetic susceptibility. Most authors propose the two-hit model as the etiology of PPCM. The “first hit” involves late-pregnancy hormonal environment that damages blood vessels, involving factors like sFlt1 and prolactin [40]. The “second hit” is an underlying susceptibility in certain women that makes them less able to tolerate this stress such as genetic predispositions, viral infections, inflammation and autoimmune reaction [4].

3.2 Prolactin and 16-kDa prolactin fragment role, Oxidative stress and endothelial dysfunction

One of the key mechanisms involves prolactin, which under conditions of increased oxidative stress undergoes proteolytic cleavage into a 16-kDa fragment. While full-length prolactin is generally associated with pro-angiogenic and immunomodulatory functions, the 16-kDa fragment displays potent anti-angiogenic and pro-apoptotic properties. This fragment has been shown to inhibit endothelial cell proliferation, migration, and survival, thereby contributing to vascular dysfunction. Importantly, the balance between prolactin and its 16-kDa derivative appears to be critical in pathological conditions such as peripartum cardiomyopathy, where increased cleavage of pro-

lactin is linked to impaired cardiac microvasculature and myocardial damage thereby promoting left ventricular systolic dysfunction [5]. Moreover, additional mechanisms involved in the pathophysiology of PPCM have been identified. Anti-VEGF factors such as sFlt-1 have demonstrated strong antiangiogenic potential in animal models, supporting the concept of a vascular basis in the pathogenesis of PPCM. The IPAC study also showed that sFlt-1 levels were highest in women presenting within 11 days postpartum and declined rapidly thereafter, supporting a temporal relationship between placental anti-angiogenic hormones and the onset of Peripartum Cardiomyopathy [6].

3.3 Genetic predisposition

Genetic susceptibility contributes to the risk of peripartum cardiomyopathy. Variants affecting sarcomeric and cytoskeletal proteins have been identified in women with PPCM [7]. Ware et al. identified rare truncating variants in genes associated with dilated cardiomyopathy in 26 of 172 women with PPCM, corresponding to 15% of the cohort. This prevalence was significantly higher than the 4.7% observed in the reference population [8,37]. TTN truncating variants were the most frequently identified variants, occurring in approximately 10% of women with PPCM compared with 1.4% of the reference population [8,43]. TTN encodes titin, an essential structural protein of the sarcomere. In a clinically characterized cohort of 83 women with PPCM, TTN truncating variants were associated with lower left ventricular ejection fraction at one year of follow up ($p = 0.005$) [8,9]. Truncating variants were also identified in other genes associated with dilated cardiomyopathy, including BAG3, LAMP2, and DMD [8].

A 2025 genetic association study found that the dilated cardiomyopathy polygenic score was associated with PPCM, with an odds ratio of 1.82 per standard deviation increase in the score (95% CI 1.43 to 2.30) [10]. In the Mass General Brigham subgroup, medical records were reviewed for 113 individuals with secondary cardiomyopathies. Among them, 66 had a high polygenic score and 7 carried a monogenic variant. This subgroup included peripartum, alcohol induced, and cancer therapy related cardiomyopathies. These proportions should therefore not be interpreted as estimates specific to PPCM [10].

3.4 Mechanism of action of bromocriptine as a prolactin inhibitor

Bromocriptine, a dopamine D2 receptor agonist, suppresses prolactin secretion from the anterior pituitary, thereby reducing circulating prolactin levels. By limiting the availability of prolactin as a substrate, it decreases cathepsin D-mediated cleavage into

the anti-angiogenic and cardiotoxic 16-kDa fragment, which is thought to play a central role in the pathophysiology of Peripartum Cardiomyopathy [11,37].

3.5 Preclinical studies

Animal models of PPCM

The STAT3 Knockout Mouse Model. An animal study conducted in 2007 by Hilfiker-Kleiner et al. in *Cell* showed that in STAT3 knockout mice, cardiac cathepsin D expression and activity was increased, leading to the generation of the cleaved 16-kDa prolactin fragment. Treatment with bromocriptine prevented the development of Peripartum Cardiomyopathy in these mice [5].

The PGC-1 α Knockout Mouse Model. In a study conducted by Patten et al. in 2012 a second vascular-hormonal mouse model was developed: cardiac-specific genetic deletion of peroxisome proliferator-activated receptor-gamma coactivator-1 α (PGC-1 α). The mechanism involved both 16-kDa prolactin activation and decreased VEGF expression. In mice lacking cardiac PGC-1 α , exogenous sFlt1 (soluble Fms-like tyrosine kinase 1, an antagonist of VEGF and placental growth factor) was sufficient to cause profound systolic dysfunction. Bromocriptine alone provided only partial reversal. Complete recovery required VEGF supplementation [12].

The PERK Signaling Model. A recent study by Shimizu et al. in *Scientific Reports* in 2021 shows that Peripartum Cardiomyopathy is not caused simply by an excess of prolactin, but rather depends on the balance between the normal 23 kDa form, which has cardioprotective properties, and the anti-angiogenic 16 kDa form [13].

3.6 Randomized clinical trials

Current scientific evidence for the use of bromocriptine in Peripartum Cardiomyopathy comes from three randomized clinical trials.

South African Pilot Study (Sliwa et al.) is the first clinical study demonstrating bromocriptine efficacy in humans with PPCM was the pilot randomized controlled trial by Sliwa et al. from South Africa, published in 2010 [14]. It compared outcomes in 20 patients: 10 received bromocriptine in addition to furosemide and ACE inhibitor, while 10 received furosemide and ACE inhibitor therapy alone. In the intervention group, bromocriptine was administered at a dose of 2.5 mg twice daily for 2 weeks, followed by 2.5 mg once daily for a further 6 weeks. At 6 months, patients treated with bromocriptine demonstrated a greater improvement in left ventricular ejection fraction (LVEF),

increasing from 27% to 58% ($p = 0.012$), compared with a more modest improvement in the control group, from 27% to 36%. The composite endpoint—defined as death, NYHA functional class III/IV, or LVEF $\leq 35\%$ at 6 months—occurred in 1 patient (10%) in the bromocriptine group versus 8 patients (80%) in the control group ($p = 0.006$) [14].

The randomized study conducted in Burkina Faso enrolled 96 women with peripartum cardiomyopathy. Of these, 48 received standard heart failure therapy consisting of furosemide and an ACE inhibitor, while 48 received the same therapy combined with bromocriptine. Bromocriptine was administered at a dose of 2.5 mg twice daily for four weeks. The bromocriptine group demonstrated greater improvement in left ventricular ejection fraction, with significantly higher LVEF values at two weeks ($p = 0.01$) and at three, six, and twelve months ($p = 0.001$). At six months, mortality was 16.6% in the bromocriptine group and 29.1% in the control group [15].

German multicenter randomized controlled trial published in 2017 by Hilfiker-Kleiner et al. In which, the efficacy of a short-term versus long-term bromocriptine regimen was evaluated as an adjunct to standard therapy in women with Peripartum Cardiomyopathy. Patients in the short-term arm received bromocriptine 2.5 mg once daily for 7 days, whereas those in the long-term arm were treated with 5 mg daily (2.5 mg twice daily) for 2 weeks, followed by 2.5 mg once daily for an additional 6 weeks. The primary endpoint was the change in left ventricular ejection fraction (Δ LVEF) from baseline to 6 months, assessed by cardiac magnetic resonance imaging. Both groups showed substantial improvement in systolic function: LVEF increased from $28 \pm 10\%$ to $49 \pm 12\%$ in the 1-week group (Δ LVEF $+21 \pm 11\%$) and from $27 \pm 10\%$ to $51 \pm 10\%$ in the 8-week group (Δ LVEF $+24 \pm 11\%$). However, there was no statistically significant difference between the two regimens ($p = 0.381$) [16]. No deaths were reported in the German study. Bromocriptine was initiated at a mean of 1.6 ± 1.6 months postpartum.

3.7 Observational studies and registries

The EORP PPCM Registry included 552 women with peripartum cardiomyopathy, of whom 85 received bromocriptine in addition to standard care. The primary composite endpoint was available for 491 women and occurred in 18 of 82 women treated with bromocriptine and standard care and in 136 of 409 women receiving standard care alone (22% versus 33%; $p = 0.044$). After multiple imputation and adjustment for confounding by indication using inverse probability weighting, bromocriptine remained associated with lower odds of the primary composite endpoint (OR 0.47, 95% CI 0.31 to

0.70; $p < 0.001$). Thromboembolic events occurred in 6.0% of the bromocriptine group and 5.6% of the standard care group ($p = 0.900$) [17].

An earlier German registry included 115 women with PPCM. During a mean follow up of six months, LVEF improved in 85% of the women, complete recovery occurred in 47%, 15% did not recover, and 2% died. A recovery rate of 96% was observed among women receiving a beta blocker, an ACE inhibitor or angiotensin receptor blocker, and bromocriptine [18]. The designs, sample sizes, comparison groups, bromocriptine regimens, and main outcomes of the clinical studies are summarized in Table 1.

Table 1. Main clinical studies evaluating bromocriptine in peripartum cardiomyopathy

STUDY	STUDY DESIGN	SAMPLE SIZE	COMPARISON GROUPS	BROMOCRIPTINE REGIMEN	MAIN RESULTS
Sliwa et al., 2010 [14]	Prospective, single-centre, randomized, open-label pilot study	20	Bromocriptine + standard heart failure therapy (n=10) vs. standard therapy alone (n=10)	2.5 mg twice daily for 2 weeks, followed by 2.5 mg once daily for 6 weeks	Bromocriptine was associated with greater improvement in LVEF at 6 months (27% to 58% vs. 27% to 36%; p=0.012). The composite poor-outcome endpoint (death, NYHA III/IV, or LVEF < 35%) occurred less frequently in the bromocriptine group; mortality was 1 vs. 4 deaths.
Yaméogo et al., 2017 [15]	Prospective, single-centre, randomized controlled study	96	Bromocriptine + standard heart failure therapy (n=48) vs. standard therapy alone (n=48)	2.5 mg twice daily for 4 weeks	At 6 months, mortality was lower with bromocriptine (16.6% vs. 29.1%; p=0.0001). LVEF improved from 37.2% to 49.9% in the bromocriptine group vs. 37.5% to 40.9% in controls (p=0.001). At 12 months, LVEF was 53.9% vs. 45.9%, respectively (p=0.001).
Hilfiker-Kleiner et al., 2017 [16]	Multicentre randomized trial	63	Short-term bromocriptine (1-week group; n=32) vs. long-term bromocriptine (8-week group; n=31); both received standard heart failure therapy	1-week: 2.5 mg/day for 7 days; 8-week: 5 mg/day for 2 weeks followed by 2.5 mg/day for 6 weeks	LVEF increased from 28±10% to 49±12% in the 1-week group and from 27±10% to 51±10% in the 8-week group. The change in LVEF did not differ significantly (+21±11% vs. +24±11%; p=0.381). Full recovery (LVEF ≥50%) occurred in 52% vs. 68%, respectively. No deaths, LVAD implantation, or transplantation occurred.

van der Meer et al., 2025 [17]	Multinational observational analysis of the EORP PPCM registry	552	Bromocriptine + standard care (n=85) vs. standard care without bromocriptine (n=467)	Regimen was not standardized and was determined by treating centres	The 6-month composite adverse maternal outcome occurred in 22.0% vs. 33.3% of patients receiving vs. not receiving bromocriptine (p=0.044). After adjustment for confounding, bromocriptine remained associated with lower odds of adverse outcome (OR 0.47, 95% CI 0.31–0.70). Thromboembolic events did not differ significantly (6.0% vs. 5.6%; p=0.900).
Haghikia et al., 2013 [18]	Prospective observational multicentre registry/cohort	115	Bromocriptine-based therapy vs. standard heart failure therapy without bromocriptine; non-randomized	2.5–5 mg/day for ≥4 weeks, in addition to standard heart failure therapy	At 6±3 months, LVEF improved in 85% of patients and full recovery occurred in 47%. A particularly high recovery rate (96%) was observed among patients receiving bromocriptine together with beta-blocker and ACE inhibitor/ARB therapy. Baseline LVEF ≤25% was associated with worse recovery.

3.8 Meta-analyses and systematic reviews

A systematic review and meta-analysis published in Heart Failure Reviews included 8 studies (2 randomized controlled trials and 6 observational studies) involving 593 PPCM patients. Bromocriptine use was associated with significantly higher survival (91.6% vs. 83.9%, RR 1.11, $p = 0.02$) and significantly higher LVEF at follow-up (53.3% vs. 41.8%, $p < 0.001$). However, there was no significant association between bromocriptine use and lower composite major adverse outcomes (13.7% vs. 33.3%, RR 0.60, $p = 0.54$) or LVEF recovery to $\geq 50\%$ (46.9% vs. 46.8%, RR 0.94, $p = 0.74$) [19]. The most recent and comprehensive meta-analysis, published in the International Journal of Cardiology in 2025, included 11 studies involving 1,706 participants. Bromocriptine was associated with a greater change in LVEF (Δ LVEF) compared to the control group, with a mean difference of 10.03% (95% CI 3.88–16.17%, $p < 0.01$). Subgroup analysis demonstrated that bromocriptine was associated with increased Δ LVEF only in patients with baseline LVEF $< 30\%$. Post-treatment LVEF was also significantly higher in the bromocriptine group (MD 8.50%, 95% CI 3.39–13.61%, $p < 0.01$). However, there was no

significant difference in LV function recovery, all-cause mortality, rehospitalization, NYHA class III/IV, major adverse cardiac events, or thromboembolism [20]. A 2024 Cochrane review on pharmacological treatment in Peripartum Cardiomyopathy concluded that evidence for bromocriptine remains of low certainty due to methodological limitations in existing reviews and trials [21].

4. DISCUSSION

4.1 Interpretation of Findings and Clinical Controversies

The dual and opposing roles of prolactin and its 16-kDa fragment highlight a complex regulatory axis with significant implications for vascular biology and disease pathogenesis [5]. Suggesting that genetic testing may play an important role in the diagnosis of PPCM, recent findings demonstrate that polygenic susceptibility may be even more prevalent than monogenic variants [10].

Despite encouraging findings, interpretation of the clinical trial data requires caution. The small sample size and open-label design of the South African trial limited the strength of its evidence [14]. Furthermore, mortality in the control group of the Burkina Faso trial was notably high (29.1%), which was substantially higher than that observed in contemporary cohorts from high-income settings, raising concerns regarding generalizability [15].

A key difference between the early African studies and the German multicenter trial concerns background therapy. The German trial used guideline directed heart failure therapy that included ACE inhibitors or ARBs, beta blockers, and MRAs, whereas furosemide and ACE inhibitors formed the basis of treatment in the earlier studies. However, the German trial had no placebo group or group receiving standard therapy without bromocriptine. Without such a control group, it is not possible to determine whether the observed benefits were attributable to bromocriptine, background therapy, or natural recovery [16].

Similarly, observational data suffer from selection bias and confounding by indication. Mixing randomized and observational data in meta-analyses introduces methodological variability [19,20,21].

4.2 Positions of scientific societies

Discrepancies between ESC and AHA guidelines highlight that the evidence regarding the use of bromocriptine in Peripartum Cardiomyopathy is still not fully established.

The ESC guidelines on cardiovascular diseases in pregnancy give bromocriptine a Class IIb recommendation in Peripartum Cardiomyopathy, indicating it may be considered as a therapeutic option. The ESC also supports the BOARD approach (Bromocriptine, Oral heart failure therapy, Anticoagulation, vasorelaxant agents, Diuretics) for acute management. In the 2025 update, bromocriptine is suggested at 2.5 mg daily for 1 week in uncomplicated cases, while a longer regimen (2.5 mg twice daily for 2 weeks followed by 2.5 mg daily for 6 weeks) is proposed for patients with severe heart failure or cardiogenic shock [2]. The guidelines further note that bromocriptine is inexpensive, generally safe, and by suppressing lactation may facilitate faster optimization of guideline-directed heart failure therapy. The 2022 AHA/ACC/HFSA Heart Failure Guidelines adopt a more cautious position on bromocriptine use in Peripartum Cardiomyopathy, stating that its efficacy and safety remain uncertain, particularly in the context of contemporary guideline-directed medical therapy and cardiogenic shock management. Unlike the ESC guidelines, no formal recommendation class is provided, although the ESC's BOARD protocol is acknowledged. The AHA/ACC/HFSA document suggests that bromocriptine may be most reasonable in patients with severe disease, such as those with LVEF <25% or cardiogenic shock, while also noting the implications of lactation suppression. It further recommends that bromocriptine, if used, should be combined with at least prophylactic anticoagulation due to potential thrombotic risk [22]. Overall, ESC guidelines are more permissive, while AHA/ACC/HFSA emphasize uncertainty and selective use. A summary of these conflicting society recommendations is provided in Table 2. The ongoing REBIRTH trial (NCT05180773) in the United States and Canada may provide the definitive evidence needed to harmonize these recommendations. The main differences between the 2025 ESC and 2022 AHA/ACC/HFSA positions on bromocriptine use in PPCM are summarized in Table 2.

Table 2. Comparison of the 2025 ESC and 2022 AHA/ACC/HFSA positions on bromocriptine use in peripartum cardiomyopathy

ASPECT	2025 ESC	2022 AHA/ACC/HFSA
Recommendation class	IIb [2]	Not assigned [22]
Protocol endorsed	BOARD [2]	Acknowledged but not endorsed [22]
Patient selection	All PPCM; longer regimen for severe cases [2]	LVEF <25% or cardiogenic shock [22]
Anticoagulation	Required (part of BOARD) [2]	At least prophylactic [22]

4.3 Bromocriptine safety concerns

The safety profile of bromocriptine in Peripartum Cardiomyopathy includes several key considerations, such as thromboembolic risk, the need for concomitant anticoagulation, suppression of lactation, and earlier concerns about rare but serious cardiovascular and neurological adverse events. Bromocriptine has been linked to an increased risk of thromboembolic complications in the postpartum period. Accordingly, the 2022 AHA/ACC/HFSA guidelines recommend at least prophylactic anticoagulation when using bromocriptine due to concerns about potential hypercoagulability [22]. Similarly, Davis et al. advise therapeutic anticoagulation alongside bromocriptine because of reported thrombotic risk [9]. In contrast, data from the EORP PPCM Registry showed comparable rates of thromboembolic events between patients treated with bromocriptine and those receiving standard care (6.0% vs. 5.6%; $p = 0.900$), suggesting that appropriate anticoagulation may mitigate this risk in Peripartum Cardiomyopathy [17]. The optimal intensity of anticoagulation with bromocriptine remains controversial. Davis et al. advocates for therapeutic anticoagulation when bromocriptine is used [9], whereas the 2022 AHA/ACC/HFSA and ESC guidelines recommend at least prophylactic dosing [2,22]. In practice, the choice is often individualized based on thromboembolic risk, degree of left ventricular dysfunction, and bleeding risk in Peripartum Cardiomyopathy. In the management of Peripartum Cardiomyopathy (PPCM), the use of bromocriptine introduces a complex clinical trade-off regarding maternal and infant health. As a potent prolactin inhibitor, bromocriptine effectively ceases milk production, necessitating the cessation of breastfeeding. This intervention must be weighed against the well-documented developmental and immunological benefits of nursing. Reflecting this tension, the AHA/ACC/HFSA guidelines underscore the importance of evaluating the “downsides of prohibiting breastfeeding” prior to the initiation of therapy [22]. Such considerations are paramount in socio-economic contexts where cultural norms prioritize breastfeeding or where the lack of access to sterile infant formula poses an independent risk to neonatal health. Historical safety concerns stem from FDA postmarketing surveillance documenting 72 seizures, 30 strokes, and 9 myocardial infarctions among postpartum women receiving bromocriptine for lactation suppression; however, the FDA states that a causal relationship has not been established. These events occurred primarily at higher doses used for lactation suppression rather than the lower doses recommended for PPCM. Contemporary PPCM trials and the EORP Registry have not replicated these concerns when bromocriptine is combined with appropriate anticoagulation.

4.4 Practical aspects of bromocriptine use

The clinical application of bromocriptine in PPCM is increasingly defined by risk stratification and hemodynamic stability. While the AHA/ACC/HFSA guidelines prioritize its use in high-risk cohorts—specifically those with an LVEF below 25% or in states of cardiogenic shock—the 2025 ESC guidelines offer a more nuanced, tiered approach. Under this Class IIb recommendation, treatment intensity is tailored to the clinical phenotype: a truncated regimen for stable presentations and prolonged therapy for those in severe acute heart failure [2,22]. Central to therapeutic success is the window of initiation; early intervention during the acute phase is thought to be most effective, as it targets the peak prolactin surge responsible for myocardial insult. Supporting this time-sensitive strategy, data from the EORP PPCM Registry suggests that adding bromocriptine to standard heart failure regimens significantly improves six-month outcomes, reducing the composite endpoint from 33% to 22% ($p = 0.044$) [17].

4.5 Dosing regimens

Two dosing regimens have been proposed based on clinical trial data.

Short-term protocol (1 week): Bromocriptine 2.5 mg once daily for 7 days. This regimen is suggested for uncomplicated cases of PPCM [23].

Long-term protocol (8 weeks): Bromocriptine 2.5 mg twice daily (5 mg/day) for 2 weeks, followed by 2.5 mg once daily for an additional 6 weeks. This regimen is recommended for patients with severe acute heart failure or cardiogenic shock [23].

The German multicenter RCT by Hilfiger-Kleiner et al. compared these two regimens and found no statistically significant difference in LVEF recovery between the 1-week and 8-week groups ($\Delta\text{LVEF} +21 \pm 11\%$ vs. $+24 \pm 11\%$; $p = 0.381$), with LVEF recovery $\geq 50\%$ achieved in 52% and 68% of patients, respectively [16].

4.6 The BOARD Protocol and Combination Therapy

This multifaceted regimen encompasses Bromocriptine, Oral heart failure therapies, Anticoagulation, Relaxing agents (vasodilators), and Diuretics. Central to this framework is the aggressive implementation of guideline-directed medical therapy (GDMT) once the patient is postpartum. This includes a foundational "four-pillar" approach: beta-blockers, renin-angiotensin system inhibitors with a preference for angiotensin receptor-neprilysin inhibitors (ARNIs), mineralocorticoid receptor antagonists (MRAs),

and SGLT2 inhibitors. Because bromocriptine therapy carries an inherent prothrombotic risk, concurrent prophylactic anticoagulation is a mandatory component of the protocol. In specific clinical scenarios, such as persistent sinus tachycardia or atrial fibrillation with rapid ventricular response, adjunctive agents like ivabradine or digoxin may be selectively employed to optimize heart rate [24,38].

4.7 Thromboembolic Prophylaxis and Anticoagulation Strategy

Given the prothrombotic nature of the peripartum period and the potential for bromocriptine to further increase the risk of vascular events [25,26,27], anticoagulation is considered a mandatory adjunct to therapy. Current AHA/ACC/HFSA guidelines advocate for at least prophylactic-dose anticoagulation throughout the duration of bromocriptine treatment. In cases of profound systolic dysfunction (LVEF <30%), this coverage should ideally be extended until six to eight weeks postpartum to navigate the window of peak thromboembolic vulnerability [22]. European Society of Cardiology (ESC) suggests a slightly broader inclusive threshold, recommending anticoagulation for patients with an LVEF <35% [2]. The intensity of anticoagulation—ranging from prophylactic to therapeutic dosing—must be tailored to the individual, balancing the severity of left ventricular impairment and baseline embolic risk against the patient's potential for hemorrhage. This cautious approach is supported by the German multicenter RCT protocol, in which all participants received low-molecular-weight heparin (LMWH) alongside bromocriptine to ensure safety [16].

4.8 Surveillance and Timing of Device Intervention

Post-diagnostic care for PPCM requires a multi-modal monitoring approach. Guideline-recommended surveillance includes serial echocardiographic assessments of biventricular function (typically at baseline, 6 weeks, 3 months, and 6 months) and the tracking of natriuretic peptides (BNP/NT-proBNP) to risk-stratify patients for future events [28]. These objective measures must be paired with frequent clinical evaluations of NYHA status and a high index of suspicion for thromboembolic complications. Furthermore, the unique recovery profile of PPCM influences the approach to electrophysiological interventions. Unlike other forms of dilated cardiomyopathy, PPCM often exhibits late-stage recovery [29]. Consequently, international guidelines advise a conservative "wait-and-see" approach for primary prevention ICD or CRT implantation, allowing for a 6-to-12-month window of optimized GDMT to determine the patient's ultimate degree of ventricular improvement [30,31].

To bridge this high-risk period without committing to a permanent implant, the wearable cardioverter-defibrillator (WCD) has emerged as a vital therapeutic tool. Research by Duncker et al. demonstrated that while roughly 12% of PPCM patients with severely reduced LVEF (LVEF <35%) experience life-threatening ventricular arrhythmias in the acute phase, many subsequently recover cardiac function. The WCD serves as a non-invasive "bridge to recovery," providing essential protection against sudden cardiac death during the initial months of peak vulnerability while allowing the necessary time for pharmacological therapies—including bromocriptine and GDMT—to facilitate myocardial healing [31,41].

4.9 Special Clinical Situations

Antepartum Presentation and Delivery. In cases of third-trimester PPCM, maternal stability is the priority. While severe hemodynamic instability or refractory volume overload necessitates immediate delivery, stable patients should be managed conservatively until at least 32 weeks' gestation to allow for antenatal corticosteroid benefits [2,22,23].

Cardiogenic Shock. For patients in cardiogenic shock, the standard approach is insufficient. These high-acuity cases require the long-term (8-week) bromocriptine protocol integrated with advanced heart failure therapies, including invasive hemodynamic monitoring and, if indicated, mechanical circulatory support (e.g., VA-ECMO or microaxial flow pumps) [2,3,22,39,42].

Subsequent Pregnancy Risks. Future pregnancy counseling must be individualized based on myocardial recovery. Women with persistent LV dysfunction face a 50% risk of further deterioration, while those with normalized function still carry a residual 20% relapse risk [32].

Lactation and Shared Decision-Making. Given that bromocriptine halts milk production, a shared decision-making model is essential for mothers wishing to nurse. In mild-to-moderate cases, clinicians may opt for a lactation-compatible regimen (e.g., metoprolol, enalapril, or spironolactone), though SGLT2 inhibitors and eplerenone must be strictly avoided due to a lack of safety data [23].

4.10 Clinical Decision-Making

Shared Decision-Making. The decision to initiate bromocriptine should incorporate patient values and preferences. Key discussion points include: uncertain efficacy in

the context of contemporary guideline-directed therapy, potential benefits of improved LVEF recovery, requirement for anticoagulation, and suppression of lactation with implications for infant feeding and maternal-infant bonding. The AHA/ACC/HFSA guidelines specifically note that "the downsides of prohibiting breastfeeding should be considered" [22].

Weighing Benefits versus Risks. For patients with mild-to-moderate PPCM (LVEF 35–45%), potential benefits may be modest given favorable prognosis with standard therapy, while costs—lactation suppression and anticoagulation—remain constant. For patients with severe PPCM (LVEF <25%) or cardiogenic shock, the potential mortality benefit may outweigh these considerations [2]. Notably, lactation suppression may paradoxically benefit some patients by enabling earlier initiation of medications contraindicated during breastfeeding, such as SGLT2 inhibitors.

Multidisciplinary Care. PPCM management requires coordinated multidisciplinary involvement including cardiology, obstetrics, maternal-fetal medicine, anesthesia, and neonatology [2]. The 2025 Lancet review recommends that larger centers establish dedicated pregnancy heart teams to ensure rapid decision-making regarding delivery timing, lactation inhibition, and mechanical circulatory support for patients in cardiogenic shock [23].

4.11 Future Research Directions

The most anticipated study is the REBIRTH trial (NCT05180773), a randomized, double-blind, placebo-controlled trial designed to enroll 200 women with PPCM in the United States and Canada. Expected to complete in 2026, it represents the first placebo-controlled trial of bromocriptine in a high-income setting with contemporary guideline-directed therapy and may resolve whether bromocriptine provides benefit beyond optimal standard care.

Predictive Biomarkers. Biomarkers in PPCM are not well established. Candidates under investigation include 16-kDa prolactin, microRNA-146a, galectin-3, and the sFlt-1/PIGF ratio [12,33,34,35]. The existence of non-responders to bromocriptine suggests alternative pathophysiological pathways in some patients, underscoring the need for biomarker-guided patient selection.

New Therapeutic Targets and Personalized Therapy. Cabergoline, with its longer half-life and convenient dosing, warrants dedicated investigation as an alternative prolactin inhibitor [36]. With approximately 15% of PPCM patients carrying pathogenic

variants in cardiomyopathy-associated genes [8], genetic profiling could inform treatment decisions—patients with TTN truncating variants may require more aggressive therapy, while those with primarily prolactin-mediated disease may derive greatest benefit from bromocriptine.

5. LIMITATIONS

This review has several limitations that should be considered when interpreting the findings. First, the review was narrative in nature, and the literature search was restricted to publications in English, which may have resulted in the omission of relevant evidence published in other languages. Furthermore, no formal methodological quality assessment or risk-of-bias assessment was performed for the included sources. The randomized studies included relatively small sample sizes, limiting the statistical power and generalizability of their findings. In addition, the German study did not include a control group of patients who did not receive bromocriptine, limiting the ability to determine whether the observed effects were attributable to bromocriptine treatment rather than to other factors or the natural course of the condition. The registry-based studies are subject to the limitations inherent to observational research, including the risk of selection bias and residual confounding. Moreover, substantial heterogeneity exists among the included studies regarding treatment regimens, background therapy, definitions and assessment of endpoints, and duration of follow-up, which limits direct comparability of their findings.

Taken together, these limitations preclude definitive causal conclusions regarding the effects of bromocriptine and do not allow categorical clinical recommendations to be made on the basis of the available evidence. The findings should therefore be interpreted cautiously and considered in the context of the methodological limitations and heterogeneity of the underlying literature.

6. CONCLUSIONS

Preclinical studies provide a biological rationale for prolactin inhibition in peripartum cardiomyopathy, but the contribution of the 16 kDa prolactin fragment to the disease in humans remains incompletely established. Evidence from randomized trials, observational studies, registries, and meta analyses suggests that bromocriptine may be

associated with greater improvement in left ventricular ejection fraction. However, consistent benefits in mortality, rehospitalization, complete ventricular recovery, and other major clinical outcomes have not been demonstrated.

Bromocriptine may therefore be considered as an adjunct to standard heart failure therapy in selected women with PPCM, particularly those with moderate or severe heart failure. The available evidence does not establish the optimal treatment duration or demonstrate superiority of the eight week regimen over the one week regimen. When bromocriptine is used, concomitant anticoagulation at a minimum prophylactic dose should be considered according to the applicable recommendations and the individual thromboembolic and bleeding risks. Suppression of lactation and its consequences for the mother and infant should be addressed through shared decision making.

Differences between the European and United States recommendations reflect the continuing uncertainty of the evidence. Adequately powered placebo controlled trials using contemporary heart failure therapy are required to determine the clinical efficacy, safety, optimal dosing, and appropriate patient selection for bromocriptine in PPCM.

DISCLOSURE

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